Radiología. 2013;55(4):294–304

RADIOLOGÍA

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UPDATE IN RADIOLOGY

Myocarditis: Magnetic resonance imaging diagnosis and follow-up☆

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Received 1 February 2012; accepted 22 July 2012

KEYWORDS
Myocarditis;
Magnetic resonance imaging;
Cardiac imaging

Abstract Myocarditis, inflammation of the myocardium, is usually due to viral infection. Diagnostic confirmation in ordinary clinical practice is difficult because the findings on the clinical history, physical examination, electrocardiogram, and laboratory tests offer scant diagnostic accuracy, and the differential diagnosis is often done with acute myocardial infarction. Cardiac magnetic resonance imaging (CMR) has become the method of choice for the diagnosis of myocarditis. In this article, we describe the CMR findings at diagnosis and during the follow-up of patients with myocarditis, the differential diagnosis with other acute processes like myocardial infarction, and the prognostic factors studied with CMR.

PALABRAS CLAVE
Miocarditis;
Resonancia magnética;
Imagen cardiaca

Miocarditis: diagnóstico y seguimiento con resonancia magnética

Resumen La miocarditis consiste en la inflamación del miocardio producida la mayoría de las veces por una infección viral. La confirmación diagnóstica en la práctica clínica habitual es difícil porque la historia clínica y la exploración física, las alteraciones en el electrocardiograma, la determinación de las enzimas cardiacas y el ecocardiograma ofrecen escasa precisión diagnóstica, y no es infrecuente que se plantee el diagnóstico diferencial con el infarto agudo de miocardio. La resonancia magnética (RM) cardiaca se ha convertido en el método de imagen de elección para el diagnóstico de la miocarditis. En este trabajo se describen los hallazgos de imagen en la RM en el momento del diagnóstico y en el seguimiento de los pacientes con miocarditis, el diagnóstico diferencial con otros procesos agudos como el infarto de miocardio, y los factores pronósticos estudiados mediante RM.

☆ Please cite this article as: Bustos García de Castro A, et al. Miocarditis: diagnóstico y seguimiento con resonancia magnética. Radiología. 2013;55:294–304.
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2173-5107/$ - see front matter

Introduction

Myocarditis is defined as acute or chronic inflammation of myocardial tissue, whether focal or diffuse, which affects any heart chamber. It was included among acquired-cause primary myocardiopathies in the classification made by the *American Heart Association* in the year 2006.[1] Later on, the European Society of Cardiology classified myocardiopathies in 2008 with a more clinical point of view grouping myocardial diseases on the basis of ventricular form and function. Chronic myocardial inflammation and/or infection may trigger a dilated myocardiopathy.[2]

If the pericardium is affected, a myopericarditis occurs, often with associated pleural and pericardial effusion. Up to 15% of acute pericarditis has important myocardial affection.[3]

It is difficult to establish its actual incidence because it is not easy to confirm diagnosis in usual clinical practice. It has been found in 5–12% of the autopsies in adults with sudden death[4,5] and in 1–9% of autopsies in general.[6] Myocarditis is the cause of 6–8% of sudden death in athletes.[7] Diagnostic confirmation is difficult because health history and physical examination, electrocardiogram (ECG) alterations, heart enzyme determination and echocardiogram are not very accurate, and it is not infrequent that differential diagnosis with acute myocardial infarction (AMI) is considered. Even though endomyocardial biopsy (EMB) is considered as the reference for myocarditis diagnosis, it is not justified in most patients. Under these circumstances, cardiac magnetic resonance (MR) imaging has become the imaging method of choice to diagnose myocarditis, especially due to its capacity for tissue characterization which enables to detect areas of edema and myocardial fibrosis.

This article describes the MR sequences used for diagnosis, the MR image findings at the moment of diagnosis and the follow-up of myocarditis patients, differential diagnosis with other acute processes such as acute myocardial infarction and the possible prognostic factors in MR studies.

Etiopathogenesis

In developed countries, viral infection is the most frequent cause. Even though the most frequent viruses during the 1980's and the 1990's were the enteroviruses (coxsackie B), in the last 10 years, other viruses such as parvovirus B19, human herpesvirus 6, adenovirus, the hepatitis C virus, echovirus, human immunodeficiency virus, the Epstein–Barr virus and cytomegalovirus, have gained importance. Other infectious agents such as bacteria, fungi, protozoan (*Trypanosoma cruzi* [*T. cruzi*], toxoplasm) and helminths may cause myocarditis. Other possible etiologies are the toxic one (anthraciclins, cyclophosphamide, traztuzumab, catecholamines), alcohol, cocaine or radiation, hypersensitivity reactions (antibiotics, diuretics such as thiazides, dobutamin, mesalazine, insect and snake bites), systemic diseases such as collagenosis, sarcoidosis, celiac disease, Wegener's disease and thyrotoxicosis.[6,8–12]

From the physiopathological point of view, myocarditis may be divided into three phases: (1) acute viral, (2) subacute immune, and (3) chronic myocardial. In the acute phase, the virus reaches the myocardium either by lymphatic or hematogenous dissemination or both in a susceptible patient and it enters the myocite. It is a short phase and it often goes unnoticed. Viral proliferation in the myocite may cause direct tissue damage, but most of the tissue damage in myocarditis occurs due to the immune system response to the virus when it reaches the myocite. The patient's evolution depends on the immune response which, in extreme cases, leads to an adverse response with autoantibody formation (antibodies anti-myosin). Viral persistence may activate the immune system continuously resulting in chronic myocardial inflammation, cardiac remodeling and function alteration, which develop a dilated myocardiopathy.[13]

In the disease's acute phase there is edema, cellular infiltrates with histiocytes and mononuclear cells, with or without myocardial cell damage. In the subacute and chronic phases, fibrosis replaces myocardial cells.[14,15]

Clinical presentation is very variable. The patient may be asymptomatic or have unspecific symptoms, suffer from chest pains, heart failure, arrhythmias, conduction alterations, cardiogenic shock or sudden death. Patients with myocarditis may present symptoms similar to those of AMI, above all the young subjects.[16] In up to 89% of myocarditis, symptoms similar to those of the influenza, gastrointestinal or urinary tract infection have been described days or weeks before. In most cases the clinical course of myocarditis is favorable and it heals without secuelae, but 5–10% of the patients present an unfavorable evolution to a dilated myocardiopathy or sudden death. In a review of 1,230 patients with myocardiopathy of initially unknown cause, 9% had myocarditis.[17] In 12% of the young patients who suffered sudden death, the cause was myocarditis.[4]

For Mahrholdt et al.[6] clinical presentation is different depending on the type of virus present in the EMB. The symptoms similar to those of an AMI are more typical in acute myocarditis by parvovirus B19. These patients often have normal systolic function and left ventricular volume and a favorable clinical course. Infection by human herpesvirus 6 and the combination parvovirus B19-herpesvirus 6 usually occurs with symptoms of recent heart failure, often with malaise and conduction alteration (bundle-branch block). It is a more insidious clinical manifestation, one with a more prolonged course and it often occurs with left ventricle systolic function alteration. Given that human herpesvirus 6 tends to remain latent after primoinfection (which usually occurs in childhood), every infected individual may suffer from reactivations throughout their lives.

Diagnosis

The health history, physical examination, the laboratory data, including heart enzymes that may rise slightly or moderately in myocarditis patients (creatinephosphokinase [CPK], MB isoenzyme of CPK, and above all, troponins T and I), and the ECG help diagnose myocarditis even though diagnostic accuracy is low. ECG may be normal or show ST alterations and T wave and Q wave alterations, auricular–ventricular blockage and branch blockage and arrhythmias such as tachycardia or ventricular fibrillation. These findings have limited value and low specificity.